UNIVERSITY
OF MICHIGAN
MAY 24 1951
MEDICAL
LIBRARY

Volume XII MAY 1951 No. 5

DISEASES OF THE NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

CONTENTS:

CLINICAL DIAGNOSIS OF PERIARTERITIS NODOSA 131
Clark H. Millikan, M.D.

GROUP PSYCHOTHERAPY BY NURSES AND ATTENDANTS 138
Rudolph Kaldeck, M.D.

RELIEF BY MYOPIA BY HYPNOSIS AND EYE TRAINING 142
Leslie M. LeCron

REGRESSIVE SHOCK THERAPY IN SCHIZOPHRENIA 147
David Rothschild, M.D., Donald J. Van Gordon, M.D., Anthony Varjabedian, M. D.

NEUROLOGICAL CLINICAL PATHOLOGICAL CONFERENCE 151

SANITARIA

ASSOCIATE EDITORS

A. E. Bennett, M.D. ..... Berkeley, California
Francis J. Braceland, M.D. ..... Rochester, Minn.
Franklin G. Ebaugh, M.D. ..... Denver, Colorado
Temple Fay, M.D. ..... Philadelphia, Pennsylvania
Milton H. Erickson, M.D. ..... Phoenix, Arizona
Hamilton Ford, M.D. ..... Galveston, Texas
Walter Freeman, M.D. ..... Washington, D.C.
George T. Harding, M.D. ..... Columbus, Ohio
A. Hauser, M.D. ..... Houston, Texas
Louis J. Karnosh, M.D. ..... Cleveland, Ohio
Frank H. Luton, M.D. ..... Nashville, Tennessee
Wm. Malamud, M.D. ..... Boston, Mass.
Karl A. Menninger, M.D. ..... Topeka, Kansas
Hans H. Reese, M.D. ..... Madison, Wisconsin
S. Bernard Wortis, M.D. ..... New York, N. Y.

Titus H. Harris, M.D.
Galveston, Texas
Editor-in-Chief

YEARLY SUBSCRIPTION $4.00
SINGLE COPIES 40 Cents
FOREIGN SUBSCRIPTION $4.50

George Biegeir, B.S., LL.B.
Chicago, Illinois
Managing Editor

Founded 1879

RING SANATORIUM

Eight Miles from Boston at an Elevation of 400 feet

For the study, care and treatment of emotional, mental, personality, and habit disorders.

All recognized psychiatric therapies are used as indicated.

Cottage accommodations meet varied individual needs. Limited facilities for the continued care of progressive disorders requiring medical, psychiatric, or neurological supervision.

Consultants in all Specialties

BENJAMIN SIMON, M. D.
Director

CHARLES E. WHITE, M. D.
LOUIS BRENNER, M. D.
FRED E. LAWRENCE, M. D.
Associates

ARLINGTON HEIGHTS, MASSACHUSETTS
Telephone AR 5-0661

FRANCIS W. RUSSELL
Executive Secretary

HARDING SANITARIUM

WORTHINGTON, OHIO

For Nervous and Mental Disorders

NINE MILES NORTH OF STATE HOUSE — COLUMBUS

HARRISON S. EVANS, M.D., Medical Director
GEORGE T. HARDING, M.D., President of Board
CHARLES L. ANDERSON, M.D., Clinical Director

L. HAROLD CAVINESS, M.D.
J. RUSSELL FRANTZ, M.D.
THEODORE J. LUKENS, M.D.
LESLIE R. GOULD, M.D.
CHARLES W. HARDING, M.D.

Telephone: Columbus FR. 2-6367

Compton Sanitarium

AND

Las Campanas Hospital

COMPTON, CALIFORNIA
(Near Los Angeles)

Compton Sanitarium is a private psychiatric hospital with 130 beds. Emphasis is given psychotherapy by a staff of resident psychiatrists. Other treatment procedures are duly evaluated and administered. Careful records are maintained. There are psychiatrically trained graduate nurses; student nurses through affiliation; nursing attendants; dietitian; occupational therapist; recreational therapist; hydrotherapist; masseuse. Las Campanas Hospital (42 beds) provides adjunct general hospital facilities. Compton Sanitarium and Las Campanas Hospital are approved by the American College of Surgeons.

G. CRESWELL BURNS, M.D., *Medical Director.*

BEVERLY HILLS CLINIC and SANITARIUM

For the Study and Treatment of Nervous and Mental Diseases

218 NO. WESTMORELAND — DALLAS 11, TEXAS

This sanitarium is a psychopathic hospital with a home-like environment and is equipped to treat psychiatric and neurological cases in a most scientific manner by means of psychotherapy, occupational therapy, recreational therapy, and physiotherapy, including insulin and electroshock treatments as well as neurosurgery. There is a trained psychologist as well as a trained social worker. An electroencephalographic instrument is available.

ARTHUR J. SCHWENKENBERG, M.D.
Medical Director

JOSEPH L. KNAPP, M.D.
Associate Psychiatrist and Clinical Director

JOHN C. POWELL, JR., M.D.
Associate Psychiatrist

ARCHIE R. FOLEY, M.D.
Senior Resident Psychiatrist

STEPHEN WEISZ, M.D.
ROBERT H. WINN, M.D.
Visiting Neuropsychiatrists

VOL. XII MAY 1951 No. 5

Published monthly by
PHYSICIANS POSTGRADUATE PRESS
277 Broadway, New York 7, New York

EDITORIAL OFFICES
UNIVERSITY OF TEXAS SCHOOL OF MEDICINE
800 Avenue B., Galveston, Texas

Entered as second-class matter at the Post Office at New York, N. Y.

DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Clinical Diagnosis of Periarteritis Nodosa

CLARK H. MILLIKAN, M.D.
Iowa City, Iowa

Since Kussmul and Maier originally described periarteritis nodosa, occasional case reports have appeared in the literature but the disease has been considered rare, as evidenced by the statement of Harris et al[1] in 1938 that there were only 300 cases of the disorder reported up to that time, and Boyd[2] in 1944 found only 95 more instances of the disorder. Interest has increased in periarteritis nodosa in the last decade and numerous articles have appeared, a few of which mention that periarteritis nodosa is not the rarity that it was once thought to be. (McCall, Marsh and Pennock.[3])

The diagnosis of periarteritis nodosa is still rarely made during the life of the patient and seldom enough is the performance of a muscle biopsy even considered. This reluctance to admit periarteritis nodosa to the realm of diagnosable entities is demonstrated by the statement of Diaz-Rivera and Miller[4] as late as 1946 when they wrote, "As evidenced by the reports of Middleton and McCarter (1935),[5] Spiegel (1936)[6] and Harris et al[1] (1939) some cases have been diagnosed before death." That there may be some change in this negative approach to clinical periarteritis nodosa is shown by the report of Logue and Mullins[7] of 11 cases about which they say, "A diagnosis was made in each instance during life. . . ." This statement can probably be taken one step further; the diagnosis can be made in many instances at the bedside, folowing a detailed history and physical examination, with as much accuracy as in cases of brain tumor or carcinoma of the stomach. Confirmation of the diagnosis by muscle biopsy may aid in convincing the skeptical.

In the literature it has been the custom to divided periarteritis nodosa into six groups: cerebral, neuromuscular, cardiac, cutaneous, gastrointestinal and pulmonary. This division into groups probably increases the difficulty of making a clinical diagnosis, rather than easing it. The implication is that periarteritis nodosa is a disease of six systems. This is not true. It is a disease of the vascular system, with both arteries and veins involved, and because it is a disease of the vascular system, any organ of the body may be affected by it. Boyd[8] stresses this point when he writes, "It should be emphasized that any artery or vein may be attacked, hence the manifestations may be confined to a single organ, an anatomical system or al vessels may be affected simultaneously or successively." The characteristic lesions of periarteritis nodosa have been described by almost every writer on the subject (references 9, 10, 11) and consist in the early stages of an acute inflammation of the vessel wall, characterized by cloudy swelling, necrosis and hyalin degeneration of the media of small and middle sized arteries. At this stage there may be many polymorphonuclear neutrophiles infiltrating the damaged areas of the vessel walls, but later lymphocytes, plasma cells and monocytes predominate. Finally, if healing does occur, there is fibroblastic infiltration of the vessel wall and marked scarring develops with narrow-

From Department of Neurology, College of Medicine, University of Iowa.

ing of the lumen. Thrombosis may be present if the vessel wall is badly damaged. Veins are involved as well as arteries, although the changes are not as striking as in the latter. Success in clinical diagnosis of this disorder is achieved when it is considered as basically one pathological entity reaching all parts of the body because of its anatomical distribution, but having its primary site only in the vascular system. When periarteritis nodosa produces symptoms referable to only one system of the body, the diagnosis has not been made (see chart—case 3) and probably will remain more or less unattainable.

Periarteritis nodosa is ordinarily a disease lasting weeks or months and there is generally ample time to obtain an accurate history and perform a careful physical examination with check-up examinations to detect any change in the physical findings. Chart X shows that periarteritis nodosa produces two classes of symptoms and signs: Generalized and localized. The general symptoms include weakness, tiredness, anorexia, weight loss and fever. Symptoms produced by local manifestations of the disease can be those referable to any organ of the body but among the more common are pain and/or numbness of extremities, with difficult use of the parts involved; headache, shortness of breath, pounding of the heart, cough, swelling of the feet and ankles, pain in the abdomen, and diarrhea. Physical examination reveals such generalized abnormal findings as diffuse weakness, fever, tachycardia, emaciation, weight loss and high blood pressure. Local abnormal physical signs include hemiparesis and aphasia (from a vascular lesion of the brain), an overactive precordium with or without cardiac enlargement and murmurs; abdominal tenderness; scattered nodules in the skin; evidence of aordiac failure (rales at the lung bases, enlargement of the liver with tenderness to palpation, and edema of the feet and ankles), areas of localized tenderness in muscle substance, and peripheral neuritis with localized weakness, sensory changes, muscle atrophy and decrease or absence of the deep reflexes innervated by the involved nerve. A characteristic feature is the changing clinical picture; fresh symptoms develop and older ones subside often to return again. Finally, routine laboratory studies generally show leukocytosis and anemia with eosinophilia aiding materially in diagnosis in 20-25 per cent of the cases. Albumin, red blood cells and casts may be present in the urine.

The cases summarized in Chart X have historical, physical and laboratory phenomena which closely parallel the reports of other writers. The unusual incidence of patients having some focus of peripheral neuritis (18 of the 20 patients reported) is in line with the impression expressed by Kernohan and Woltman[9] that neuritis, associated with panarteritis, is "much more common" than is generally realized.

Part of Table from Harris and Lynch,[1] 101 cases:

Fever	80
Leukocytosis	70
Albuminuria	65
Abdominal pain	57
Edema	52
Weight loss	48
Neuritis	48
Hypertension	46

With the picture of a disseminated disease of the vascular system in mind, it has been possible to make a bedside diagnosis of periarteritis nodosa in 16 of the 20 patients listed in Chart X. Following clinical diagnosis, an attempt was made to demonstrate the characteristic vascular lesions by means of muscle biopsy. If an area of muscle tenderness is present it is chosen as the site of the biopsy. A generous portion of muscle should be obtained and serial sections of the tissue should be studied with the greatest care. Under routine pathological examination, only 20-25 per cent of biopsies are positive for periarteritis nodosa in cases ultimately proven to have the disease. Study of serial sections of the biopsy specimen is made because it has been noted that sections from one portion of the block of tissue may show periarteritis, while other sections from the same block are entirely normal. This was true in case six. It will be noted that case eight had a negative biopsy but the autopsy showed the existence of severe periarteritis. Case nine had one negative biopsy (calf muscle), one positive biopsy (vessel from abdominal wall) and the autopsy re-

vealed widespread lesions of the disease. All of the cases here reported have had the diagnosis confirmed by biopsy or autopsy; however, a number of patients have been diagnosed periarteritis nodosa who have had negative biopsies. This is to be expected, as the chance of obtaining tissue containing involved vessels is roughly one in four.

Case Histories

D. L. K. was a 49 year old white woman first seen at the University Hospitals in June, 1941, at which time she complained of having had numb fingers and toes, off and on, for the past three years. She had received liver therapy since November, 1940, without relief of any of her symptoms. Physical examination at that time was negative, except for the absence of pallesthesia on the left great toe. All of the blood findings were within normal limits. The diagnosis was deferred.

In June 1942, the patient returned with the same complaints and in addition stated that her fingers would blanch on immersion in cold water. She tired easily and had given up golf because she could not walk more than two blocks without becoming exhausted. A week before this visit she developed a sudden sharp pain in her outer right shin. This area was extremely tender to touch, but improved rapidly. Physical examination was negative except for diminished pallesthesia on the lower extremities, with more involvement on the left than on the right. No diagnosis was made.

The patient was admitted to the hospital on October 26, 1942, with additional complaints of poor use of the right hand and shortness of breath.

Physical examination revealed the following positive signs: pulse rate 128, blood pressure 190/130, enlarged heart, paralysis of flexors of the fingers of the right hand, paresis of the flexors of the right elbow, sensory less on the palmar surface of the right hand, spotty areas of sensory impairment on the ventral surface of the left forearm, impaired deep sensation over both lower extremities to the knees, biceps jerk absent on the right and normal on the left, absent deep reflexes in the lower extremities and absence of response to faradic stimulation over all of the involved muscles. Urinalysis, differential smear of the blood, stool examination, blood serology and spinal fluid examinations were all normal. The red blood count was 3,000,000. Following admission to the hospital, the patient complained of a great deal of pain in the back and shoulders which was relieved to some extent by the application of heat. The cardiac rate continued rapid. Cardiac failure intervened, and despite energetic treatment, this syndrome persisted. Uncontrollable diarrhea appeared and on the 23rd day of her hospital stay there was a marked increase in the respiratory rate and temperature, together with the appearance of pain in the left chest. Examination at that time revealed a harsh pleural friction rub over the left lower lateral chest wall and later a precordial friction rub. The body temperature was 103° F. and this elevation persisted, in spite of sulfadiazine therapy, until the time of the patient's death on the 29th hospital day. Autopsy revealed widespread periarteritis nodosa.

This patient had the usual weakness and easy fatigability commonly seen even in the initial phases of periarteritis nodosa. There came symptoms and physical signs of a widely disseminated disorder: Rapid pulse, arterial hypertension, enlarged heart and asymmetrical polyneuritis. This widespread pathology with the slowly progressive course suggested the diagnosis of periarteritis nodosa. As is often true cardiac failure appeared and persisted in spite of treatment.

CASE VIII C. C., a 71 year old white man, was admitted to the hospital on May 6, 1945, complaining of swelling of the feet and ankles for seven weeks, afternoon temperature elevation for three to four weeks, weight loss, weakness and numbness of the right hand for two days. Preceeding the onset of his illness he had a severe cold, but other than this, his health had been good.

Physical examination revealed the following positive findings: Blood pressure 160/80, weak legs with flabby calves—worse on the right, weak opposition of the thumb and little finger of the right hand, absent deep reflexes throughout, swollen feet and ankles, deep sensory impairment on the shins and feet.

Laboratory examination: Hemoglobin 9.5 grams, red blood count 3,100,000, white blood count 20,000 and blood smear normal. Numerous casts and red blood cells were found in the urine. The blood urea nitrogen was 14.0 milligrams per cent and the creatinine 1.0 milligrams per cent. Agglutinations for typhoid and malta fever were negative. A muscle biopsy from the calf was normal. The blood and spinal fluid serologies were negative.

Clinical diagnosis: Periarteritis nodosa.

From May 10, 1945 to May 29, 1945, the patient was given 15,000 units of penicillin every three hours intramuscularly. From May 30, 1945 to June 5, 1945, he was given 20,000 units of penicillin five times a day. The total dose was 3,035,000 units.

During his hospital stay there was gradual change in the blood pressure until it reached a consistent figure of 180/100. There was temperature elevation, and on June 9, 1945, the patient went into acute cardiac failure from which he did not recover. He expired on June 10, 1945.

Autopsy showed periarteritis in all stages of development.

This is another patient with generalized abnormal physical signs (fever, weakness, anemia, and weight loss) and an asymmetrical polyneuritis with casts and red blood cells in the urine. The muscle biopsy from the calf was normal but the autopsy showed the periarteritis which has been clinically diagnosed. Acute cardiac failure again was the terminal process.

CASE III J. H., a 46 year old man was first seen on October 5, 1943, complaining of pain in the left side of his head for six weeks, headache off and on for six months, generalized weakness for six months. The history showed that the patient had been well until he began to have an occasional generalized headache six months before the examination. The complaint had gradually increased in severity until the patient had to stop work six weeks before admission. The remainder of the history was essentially negative. Physical examination showed nothing unusual.

The laboratory examination was negative except for a spinal fluid cell count of 130 lymphocytes per cubic millimiter of fluid.

The diagnosis was deferred after the return of negative agglutination tests for Brucellosis, and cultures were negative for torula histolytica. The patient was discharged on symptomatic medication.

He returned to the hospital on May 3, 1944. During the interval, he had returned to work until a month before his second visit to the hospital, at which time the pain in the head became more severe. Ten days before his return, his headache became very severe, kept him awake at night and ultimately prompted his return to the hospital.

Physical examination was again negative. A repeat spinal fluid examination showed the presence of 66 lymphocytes per cubic millimeter of fluid. All special studies were negative. The patient returned

CHART X

		Name	Age	Sex	Generalized Symptoms: Weakness	Tiredness	Weight loss	Fever	Focal Symptoms: Weak legs	Pain in legs	Numbness in legs	Pain in upper extremities	Weakness in uppers	Numbness in uppers	Pain in abdomen	Headache	Generalized Signs: Fever	Tachycardia	Hypertension	Weakness	Weight loss	Focal Signs: Neuritis	Localized tenderness	Localized weakness	Localized atrophy	Sensory impairment	Absent deep reflexes	Cardiac failure	Peripheral vascular occlusion	Abdominal tenderness	Focal brain damage (aphasia, hemiplegia)	Papilledema	Erythematous nodules	Laboratory Findings: Anemia	Leukocytosis	Eosinophilia	Albuminuria	Hematuria	Nitrogen retention	Clinical diagnosis	Biopsy	Died	Autopsy
1.	W.S.	46	M		+	+					+																																
2.	D.K.	49	F		+	+					+																																
3.	J.H.	46	M													+																											
4.	W.S.	62	M		+	+	+	+																																			
5.	H.E.	54	M		+	+		+			+		+																														
6.	L.N.	23	M		+	+	+	+					+																														
7.	S.D.	72	M		+	+					+																																
8.	C.C.	71	M		+	+					+																																
9.	H.C.	51	M		+	+	+						+																														
10.	C.L.	54	M		+	+																																				
11.	M.C.	38	F		+	+		+																																			
12.	J.D.	53	F		+	+					+																																
13.	G.C.	32	M		+	+																																				
14.	E.S.	60	M		+	+																																				
15.	M.B.	41	F		+	+																																				
16.	R.A.	60	M		+	+																																				
17.	P.S.	65	M		+	+																																				
18.	W.D.	74	M		+	+																																				
19.	A.C.	49	F		+	+																																				
20.	J.K.	30	F		+	+																																				

home, having been given instructions to return to the hospital should anything unusual occur.

The patient was seen again on June 2, 1944, at which time he again had severe headache. On May 31, 1944, he first noted numbness of the left hand, face and leg while eating, and that he was poorly able to control his movements during that time. The symptoms disappeared only to return at intervals, without however, producing any lasting disability. Physical examination was entirely negative, except for the presence of a partial left homonymous hemianopsia which was confirmed by the Department of Ophthalmology.

All laboratory tests were negative. The spinal puncture was not repeated.

A tentative diagnosis of focal disease of the right side of the brain, probably neoplastic, was made. On June 9, 1944, an air ventriculogram was done which was interpreted as showing an expanding lesion in the right temporal area.

Surgical exploration on June 9, 1944, was described in part as follows: "The brain was under moderately increased tension, the convolutions being somewhat flattened, and the sulci more shallow than normal. In the anterioinferior portion of the parietal lobe, the cortex appeared abnormal. The vessels appeared to be obliterated with scattered small white areas of what appeared to be scarring. This gave the vessels a beaded appearance. The surface of the brain itself in places had a grayish-white appearance and was somewhat more firm than the surrounding brain. A small area in this region was removed for biopsy examination."

Following the operative procedure the patient had a severe left hemiplegia which showed considerable improvement at the time of discharge from the hospital on June 17, 1944.

The patient returned for a check-up examination on July 21, 1944. During the interval he had improved gradually. Physical examination revealed the following positive signs: Moderately severe left hemiparesis, almost complete loss of deep sensation on the left side of the body, left homonymous hemianopsia, a slow difficult hemiparetic gait and one plus mental retardation.

Again, all laboratory examinations were negative including spinal fluid cell count. As the patient had no particular complaints, symptomatic therapy for the hemiparesis was prescribed, and he returned home.

On October 13, 1944, he again entered the hospital. On October 12, 1944, he had gotten up in the morning practically unable to talk. His speech became worse during that day. There was moderate difficulty in swallowing but there was no regurgitation through the nose of fluid. The positive physical signs were: Moderate left hemiparesis, left homonymous hemianopsia, moderately severe loss of deep sensation on the left side of the body and three to four plus aphasia, and considerable difficulty in swallowing. Laboratory examination was negative. He was returned to his local hospital for care.

On January 24, 1945, he again returned. During the interval his speech had improved markedly and the difficulty in swallowing had disappeared. His general health showed nothing unusual. The positive findings were: One plus left hemiparesis, one plus impaired deep sensation on the left leg and hand, and speech one plus slow.

This detailed case history is included to record the diagnostic difficulties present when the symptoms and abnormal physical signs, produced by the vascular lesions, are located only in one portion of the body, and when none of the generalized phenomena emphasized above are present. Were such a case observed over a long time period periarteritis nodosa could be included in the differential diagnosis, but such a clinical diagnosis would not be tenable without subsidiary evidence.

Differential Diagnosis

Because of the often widespread involvement of the vascular system, panarteritis can produce impaired function of any organ of the body. It is impossible to list an "exact differential diagnosis." The disease has been mistaken for myositis, trichinosis, amebiasis, tuberculosis, syphilis, brain tumor, cardiovascular hypertensive disease, nephritis, acute intra-abdominal conditions, lead neuritis, alcoholic polyneuritis, and a host of others. This confusion is easy to understand, as panarteritis can produce the symptoms of any of these diseases and, as previously mentioned, when the arteritis produces its manifestations in only one portion of the body, a clinical diagnosis is well nigh impossible. However, it more commonly produces widespread damage, and therefore more symptoms and signs, than do many of the disorders listed. In such patients, the history, physical examination and laboratory studies can be used to eliminate syphilis, tuberculosis, trichinosis, amebiasis, Hodgkin's disease, malta fever and similar diseases producing widespread dysfunction from the diagnosis. If, after such a process of exclusions, the patient still has evidence of a chronic illness, with generalized and focal symptoms and generalized and focal signs involving more than one "system" of the body, and with either anemia, leukocytosis, eosinophilia, albuminuria or hematuria, a clinical diagnosis of panarteritis can be made. Biopsy of muscle substance may sustain such a clinical impression.

Etiology

The etiology of periarteritis is unknown. A filterable virus,[12] toxic reactions,[13] syphilis, and streptococcal infection have been suggested as etiologic factors in the past, but have been largely discarded because of inadequate evidence to substantiate them. Zeek[14] feels that hypertension itself may cause the arterial changes; however, the lesions are often present without hypertension. The association of periarteritis with rheumatic fever, disseminated lupus erythematosis,[15] dermatomyositis, and scleroderma has caused comment but no common

etiologic agent has been found. By administering desoxycorticosterone acetate to rats, Selye[16] has produced lesions similar to those of periarteritis nodosa, which may suggest the presence of a hormonal imbalance.

Wilson and Alexander[17] report that in 300 cases of periarteritis nodosa, bronchial asthma was identified in 54, or 18 per cent. This, together with the occurrence of eosinophilia in 20-25 per cent of periarteritis cases, has suggested that some type of sensitivity may play a role in producing the lesions. Rich[18] records five cases given serum therapy with ultimate autopsy examination and demonstration of vascular lesions characteristic of periarteritis nodosa. Whether the serum therapy, or the disease for which it was given, or other factors produced the lesions cannot be determined, although Rich states that it "is persuasive evidence that vascular lesions of this type can be a result of the anaphylactic type of hypersensitivity reaction which is responsible for serum sickness." Four patients, who had experienced serum sickness, were studied at autopsy by Clark and Kaplan.[19] Two patients showed lesions of periarteritis nodosa.

Rich[20] has also reported the production of periarteritic lesions in rabbits by sensitizing them to horse serum. He concludes from this work that periarteritis is produced by vascular response to some antigenic agent. No quantitative studies of antibody formation were done. Hopps and Wissler[21] have confirmed Rich's observations and did antibody studies, but admit that there was a lack of quantitative correlation between the degree of pathology and the amount of circulating antigen.

In contrast to these reports Smith and Zeek[22] state that, "no lesions were found in five rabbits which had been made hypersensitive to sterile horse serum."

At the State University of Iowa an attempt has been made to produce the lesions of periarteritis in the rabbit by the technique described by Rich and Gregory,[20] which consisted of sensitizing the animals to horse serum and studying tissue sections either after death had occurred from the effects of the serum injections, or the animals had been sacrificed. Twenty albino rabbits were used in the initial experiments. In nine animals the original protocol described by Rich and Gregory[20] was followed exactly while in eleven rabbits, variations of dosage were used, mostly tending toward larger amounts of horse serum than that used by Rich and Gregory.[20] Skin reactions, indicating the production of sensitivity to the serum, were noted in all animals, in varying degrees of severity. Ultimately, multiple tissue sections from brain, meninges, heart, lungs, major and moderate sized blood vessels, pancreas, mesentery, kidneys, adrenals peripheral nerves, diaphragm, abdominal muscles and leg muscles were studied for gross and microscopic evidence of panarteritis. No evidence of lesions was found.

Twenty additional rabbits were then subjected to intravenous injections of horse serum with different brands of horse sera being used, and several other dosage schedules followed. Although positive skin tests revealed evidence of the development of sensitivity to the horse serum, prolonged study of many tissue sections from the above mentioned portions of each animal yielded no evidence of arteritis.

No positive conclusions can be made from such observations. The etiology of periarteritis nodosa is not known, although some evidence has been presented to document the assumption that the lesions are produced by a reaction to an antigen. This possibility has been of no value in the clinical management of the 20 cases of periarteritis nodosa reviewed in Chart X. A careful search was made in each instance for a possible antigen which might act as an inciting factor in the production of the lesions but none was found. It is possible that there may be multiple causes for the lesions seen in this disorder, or that it is not a primary disease at all, but only the appearance in the vascular system of lesions, the cause of which is to be found elsewhere.

Treatment

There is no specific treatment for periarteritis nodosa. McCall, March and Pennock[3] state that sulfonamide was used in seven patients without altering the course of the disease. Goldman and Dickens[23] report the apparent cure of periarteritis nodosa with

sulfapyridine while Rich reports that periarteritis developed following the ingestion of sulfathiazol, and assumes that the drug is the agent probably responsible for the occurrence of the lesions.

Penicillin has been used in several patients in this series but has not apparently altered the course of the disorder. Benadryl and pyribenzamine have not produced improvement and while aminophyllin, nicotinic acid and papaverine hydrochloride may be useful in controlling peripheral vascular spasm, there is no evidence that the natural course of the disorder is changed.

It is necessary that these chronically ill patients receive adequate vitamin and caloric intakes and whatever symptomatic measures may make them more comfortable. Aspirin and/or codeine will ordinarily control the discomfort. Physiotherapy, as it can be tolerated, should be used on the involved arms and legs to prevent contractures and keep what potential function remains at its maximum level. Care of the skin is important as decubiti develop rapidly and surgical procedures may be necessary if mesenteric thrombosis or other thrombotic complications occur.

Prognosis

The literature concerning periarteritis has grown around descriptions of patients coming to autopsy, seldom clinically diagnosed before death; hence the general impression has been that this disorder almost always is a fatal one. Whether many unrecognized cases of arteritis exist which do not end fatally and may actually recover, cannot be determined at the present time. Twelve of the twenty patients here reported are alive, and six of these are clinically well and working. These living patients have been followed a minimum of two years since their acute illness. It is, of course, possible that there will be recurrence or development of new vascular lesions, and that these patients will ultimately die of panarteritis or of damage produced by it. It is possible, however, that as diagnostic accuracy increases, it will become evident that the process may rather frequently stop short of causing death. It has been impossible in many instances in this group of patients to prognosticate with accuracy the outcome of the clinical course, except in those individuals developing cardiac failure or coronary thrombosis or both. Only one of eight such patients is still alive. Patient No. 6 was so ill that death seemed imminent on three occasions but clinical recovery was ultimately complete, and the patient has now been working steadily for four years. That the disorder is a serious one seems certain, and the prognosis, on the basis of our present knowledge, is poor.

Conclusion

A clinical diagnosis of periarteritis nodosa can be made with accuracy in a considerable number of the patients having the disease, by using the criteria recorded above.

Many patients having periarteritis nodosa have involvement of peripheral nerves.

The etiology of this disorder is unknown and there is no specific treatment for it.

Although the prognosis in the past has been bad, this concept will probably change toward a moderately more favorable view as greater accuracy in the diagnosis of periarteritis nodosa is attained, and the diagnosis is made long before the patient dies.

BIBLIOGRAPHY

1. Harris, A. W., Lynch, G. W., and O'Hare, J. P.: Periarteritis Nodosa. *Arch. Int. Med.* 63:1163-1182 (June) 1939.
2. Boyd, Linn J.: Clinical Aspects of Periarteritis Nodosa. *Bull. N. Y. Med. College,* Flower and Fifth Ave. Hospitals, 1:219-226 (Dec.) 1938.
3. McCall, M., and Pennock, J. W.: Periarteritis Nodosa: Our Present Knowledge of the Disease. *Ann. Int. Med.* 21:628-637 (Oct.) 1944.
4. Diaz-Rivera, R. S., and Miller, A J.: Periarteritis Nodosa: A Clinico-pathological Analysis of Seven Cases. *Ann. Int. Med.* 24:420-443 (Mar.) 1946.
5. Middleton, M. S., and McCarter, J. D.: Diagnosis of Periarteritis Nodosa. *Am. J. M. Sc.* 190: 291-308 (Sept.) 1935.
6. Spiegel, R.: Clinical Aspects of Periarteritis Nodosa. *Arch. Int. Med.* 58:933-1040 (Dec.) 1936.
7. Logne, R., and Mullins, F.: Polyarteritis Nodosa: Report of Eleven Cases With Review of Recent Literature. *Ann. Int. Med.* 24:11-27 (Jan.) 1946.
8. Boyd, Linn J.: The Clinical Aspects of Periarteritis Nodosa. *Bull. N. Y. Med. College,* Flower and Fifth Ave. Hospitals, 3:32-41 (Apr.) 1940.
9. Kernohan, James W., and Woltman, Henry W.: Periarteritis Nodosa. *Arch. Neurol. and Psych.* 39:655-686 (Apr.) 1938.

10. Arkin, Aaron: A Clinical and Pathological Study of Periarteritis Nodosa. *Am. J. of Path.* 6:401-426 (July) 1930.
11. Malamud, N., and Foster, D. Bernard: Periarteritis Nodosa. *Arch. Neurol. and Psych.* 47:828-838 (May) 1942.
12. Harris, W. H.: Etiology and Pathology of Periarteritis Nodosa. *South. Med. J.* 19:426-430 (June) 1926.
13. Helpern, M., and Trubek, M.: Necrotizing Arteritis and Subacute Glomerulonephritis in Gonococcus Endocarditis. *Arch. Path.* 15:35-50 (Jan.) 1933.
14. Higgins, W. H.: Periarteritis Nodosa; Clinical Manifestations and Postmortem Findings with Report of Six Cases. *South. Med. J.* 39:453-460 (June) 1946, Discussion by Pearl Zeek.
15. Banks, B. M.: Is There a Common Denominator in Soleroderma, Dermatomyositis, Disseminated Lupus Erythematosus, the Libman-Sacks Syndrome and Polyarteritis Nodosa? *New England J. Med.* 235:433-444 (Sept. 18) 1941.
16. Selze, Hans: Hormonal Production of Arthritis. *J.A.M.A.* 114:201-207 (Jan. 22) 1944.
17. Wilson, Keith S., and Alexander, Harry L.: The Relationship of Periarteritis Nodosa to Bronchial Asthma and Oother Forms of Human Hypersensitivity. *J. Lab. and Clin. Med.* 30:195-203 (Mar.) 1945.
18. Rich, Arnold: The Role of Hypersensitivity in Periarteritis Nodosa. *Bull. Johns Hopkins Hosp.* 71:123-141 (Sept.) 1942.
19. Clark, E., and Kaplan, B. I.: Endocardial, Arterial and Other Mesenchymal Alterations Associated with Serum Disease in Man. *Arch. Path.* 34:458-475 (Oct.) 1937.
20. Rich, A. R., and Gregory, John E.: The Experimental Demonstration That Periarteritis Nodosa is a Manifestation of Hypersensitivity. *Bull. Johns Hopkins Hosp.* 72:65-68 (Feb.) 1943.
21. Hopps, H. C., and Wissler, R. W.: The Experimental Production of Generalized Arteritis and Periarteritis. *J. Lab. and Clin. Med.* 31:939-957 (Sept.) 1946.
22. Smith, C. C., and Zeek, Pearl M.: The Role of Various Factors in the Etiology of Periarteritis Nodosa in Experimental Animals. *Am. J. Path.* 33:147-157 (Jan.) 1947.
23. Goldman, B. A., Dickens, K. L., and Schenken, J. R.: The Apparent Cure of Periarteritis Nodosa with Sulfapyridine. *Am. J. M. Sc.*, 204: 443-447 (Sept.) 1942.

Group Psychotherapy By Nurses and Attendants

RUDOLPH KALDECK, M.D.*
Harding, Massachusetts

The purpose of this report is to demonstrate the use of nurses and attendants as group leaders in a large group psychotherapy program conducted in a mental hospital with minimum of trained psychotherapists. It should be emphasized from the beginning that such a project can be successful only if the group leaders have been carefully selected and given proper instruction. It is of paramount importance that they be continuously supervised in their work by a psychiatrist with experience in group psychotherapy. However, it is of little value to describe the scholastic background and the extensive training we would like the group leaders to have, when persons who meet these ideal demands at the present time are not available in our mental hospitals but the need for psychotherapy is great.

In a paper on qualifications and training of group therapists S. R. Slavson[1] stated that "No one who has not had a thorough and prolonged experience in general psychotherapy ought to attempt to do group therapy in any of its forms." There is no objection to this statement as far as group therapists are concerned who are supposed to carry out their work independently. In our project the group leaders meet every week with the author to discuss both general aspects of their work and problems of individual patients. These seminars with the nurses and attendants participating in group therapy form an important part of the project and orient the group leaders to work under guidance and supervision.

So many activities in the treatment of psychoses have been called group therapy that it is necessary to define what concept was used in our project. Gifford and MacKenzie[2] reviewed the literature and stated that the various types of group therapy of psychoses can roughly be classified as a) repressive-inspirational, b) didactic, and c) dynamically oriented methods of approach. In the practical work with patients often

*Director of Clinical Psychiatry, Medfield State Hospital.

combinations of these methods are used. With the majority of our group leaders being attendants there was no point in lengthy discussions of the theory of approach. In the practical work however, it was possible to discourage repressive tendencies of the group leaders and to keep any didactic approach at a minimum. Although the leaders were mostly not aware of it, they assumed the role of a parent figure and the other members of the group acted as sibling substitutes, in this way re-enacting the patients' early, emotionally charged experiences. The majority of our hospital population are schizophrenics, who have a great tendency to withdraw into isolation. The work in the group encourages them to re-establish human contacts and enables them to talk without being rebuked. For these reasons group therapy of psychotics should not be considered just as the second best because not enough personnel is available to do individual therapy. Klapman[3] has pointed out that patients with whom a positive transference can be established only after months of individual treatment, if at all, may be able to overcome their resistance under the influence of a group. My own concept of group therapy with psychotics has been clarified by my work as a member of the staff of the Boston State Hospital and by participation in discussions conducted by Elvin V. Semrad.[4] I wish to quote the latter's statement about group therapy:

"Our group technique is essentially a 'participation technique' in which the physician plays the role of a 'catalyst,' participating freely in casual conversation on any subject the group may wish to bring up. He does not hold himself to a questioning role but does . . . whatever seems appropriate to keep up a free and easy conversation."

Our project of group psychotherapy conducted by nurses and attendants was started at the Medfield State Hospital in the Fall of 1949 when only a small number of physicians was available for psychiatric work at the hospital. The doctors could not be burdened with any additional responsibility. Although the nursing service had their own problems due to the insufficient number of personnel it was obvious that they were the only possible source of recruitment of group leaders. The superintendent of the hospital, Dr. Theodore Lindberg, encouraged and promoted our project and the director of the nursing service, Mrs. Eleanor Loija, R.N., gave it her full support. At a meeting of the nurses and attendants the general idea of the project was explained; it met with a most gratifying interest. Only a few attendants and nurses could be selected for active participation in order not to interfere with the other activities in the hospital. The selection was made on the basis of availability, experience in dealing with psychotic patients, and emotional maturity.

Those who could be expected to become good leaders of therapeutic groups were given a series of lectures in which the purpose and the technique of group therapy were described in simple terms. The therapeutic purpose of the hospital was discussed and it was pointed out that it was not enough to keep the patients out of major trouble. It was mentioned in what way the patients could be helped to get along with other people and what the meaning of group therapy was among the other methods available for treatment. The importance of bringing patients together and inducing them to talk of their difficulties was stressed. In this way the patients might find out that other people had problems similar to theirs. By conducting the group meetings with this purpose in mind and by showing sympathetic understanding and honest interest, the group leaders can help the patients to overcome their feeling of loneliness and their tendency to withdraw from contacts with other people.

At this point the prospective group leaders expressed anxiety as to their qualifications for such a task. It must be admitted that the author entertained serious doubts himself as to how such a project would work out. The attendants were reassured that their practical experience in dealing with psychotic patients was a great asset. It was emphasized that an attitude of helpfulness and sincere interest was more significant than technical knowledge.

The organization and technique of group therapy was discussed in following sessions. Groups of ten to fifteen patients would be selected by the ward physicians. Wishes expressed by the patients and recommendations made by the group leaders would be

considered. Active phychotic behavior and long duration of the illness should not exclude any patient from participation in the group. The great need of patients on the "back wards" for this kind of treatment was emphasized and the poor foundation of the concept of so-called deterioration was explained. Hourly group meetings should be held three times a week. Regular and punctual attendance by the leaders is vital. The patients should not be forced to come to the sessions, but should be invited and persuaded. The leader should be a member of the group. He should not be the school teacher sitting over his pupils; leader and patients should gather in an informal circle. At the beginning the group leader should participate in casual conversation and leave it to the group to change the subject if a topic brought up by one patient should not interest the others. In many of these recommendations the author utilized the experiences at the Boston State Hospital (James Mann and Harold Mann[5]).

In later sessions which were held with the prospective group leaders they assumed much more active roles by asking pertinent questions and discussing the project from different angles. More detailed instructions as to the technique are given. The first session with the patients may be started with a brief explanation of the purpose of the therapy by the leader: that the purpose of the meetings was to help one another and that the members of the group could talk about whatever they wanted. Should one of the patients begin a conversation there would be no need for the therapist to give even a brief orientation. The group leaders were warned not to make speeches and not to show off with their own knowledge. The importance of a permissive attitude was stressed so that the patients would feel free to express themselves. A patient should not be rebuked when criticizing the hospital even in a hostile way or when revealing sexual feelings with profane expressions. The prospective group leaders were told that in the first meetings many patients would test out the situation and ask for favors such as being placed on parole, transferred to a better ward or released from the hospital. The group leaders should listen to the patients with friendly interest but make it clear that they had no authority to make administrative decisions. Attention was directed to close observation of the patients' actual behavior. A member of the group rushing into the room may indicate his competition with the other patients and reenact early experiences of rivalry with his brothers and sisters; other non-verbal cues were discussed. Finally it was pointed out to the group leaders that many more problems would come up during the actual work; they would meet once a week with the supervising physician; in these meetings they would report about experiences and difficulties and would receive support and instruction in a free discussion.

The first seven groups were activated in November 1949. Since then new groups have been added. During the last year more physicians have joined the medical staff of the hospital and the psychology department has expanded; several attendants started additional groups. At present twenty-one groups, each with twelve to fifteen patients are functioning. Three groups are conducted by physicians, three by psychologists, one by a social worker, two by nurses and twelve by attendants. In accordance with the purpose of this report only our experiences with the groups conducted by nurses and attendants will be described. Although a certain turn-over among the attendants was anticipated, it is gratifying to report that six of the original seven groups still have the same group leaders. As planned in advance, each group meets three times a week and the group leaders meet once a week with the author.

I do not hesitate to state that these seminars with the group leaders have been most rewarding. It has been fascinating to observe their growth and development stimulated by their actual work. At the onset they were mainly interested in altering reality factors: they wanted a patient's relatives to visit him more often or to have better equipment for the activities on the ward. They might suggest having a patient assigned to more suitable work, or transferred to another ward. These recommendations were followed up by the hospital physicians in order not to discredit the at-

tendants. However, they were made to understand that the purpose of group therapy was something else. The attendants soon ceased to favor those patients who praised them and to resent the abusive and overcritical attitude of other patients.

One female group leader reported about a woman patient who soiled her clothes with food, crawled along on the floor and talked in a high-pitched voice; this attendant added that Rose (the patient) obviously wanted to be a baby and the other patients in the group commented about her behavior in this sense. At the next seminar with the group leaders the same attendant reported that Rose, when spoken to, withdrew into the far corner of the room and finally made an attempt to climb into an empty, unused fireplace. The physician commented that Rose must have been scared away from contacts with other people and that she was looking for the protected life of a baby. Perhaps her climbing into the fireplace expressed a desire to return to the complete protection of the unborn baby. This interpretation was readily accepted and the attendant was able to make appropriate comments in following sessions with her group.

Another attendant, a man, discussed a male patient who spoke continuously and disparagingly about his sister claiming she always tried to steal his things. At another session that patient yelled at his fellow patients demanding that they stop calling him a woman. Suddenly he became very excited, dropped his trousers and exposed his body to the group. The attendant was asked what he did in this situation and replied that he calmly told the patient that the group knew he was a man, a strong man, and there was no need to show it. The patient calmed down immediately and remained fully dressed. Many more instances of similar situations could be added. They demonstrate that the attendants learned to accept the patients' disturbed behavior and to make appropriate comments. They became increasingly aware that the seemingly senseless behavior of the psychotic had a definite meaning, although it may not be immediately understandable. The attendants learned to observe the patients with new interest and increased respect.

There are some groups conducted for a particular character of patient: for the postlobotomy patients, for those undergoing insulin coma treatment, and a group for alcoholics, conducted by an attendant who is a member of the Alcoholics Anonymous. These groups are not described in detail.

It is not intended to attempt a statistical evaluation of our project at this time except to state that our experience with group psychotherapy conducted by attendants and nurses has been extremely satisfactory. Lately our psychology department started to score the condition of patients in group therapy by means of Malamud and Sands[6] rating scale. It is intended to repeat the rating at intervals of several months and to report on the results at a later time. Even without statistical documentation the beneficial effect on the patients is demonstrated by their better behavior. They utilize the opportunities presented at the meetings to talk freely without being reprimanded and to compare their own problems with those of other patients. They learn to verbalize their anxieties instead of acting them out and are induced to do business with other people at the proper level. Three patients, attending the groups, who had been in the hospital for many years have since left on indefinite visit. Several others have shown so much improvement that they are able to go out on occasional visits to their families. Others could be transferred to better wards. Only very few patients, notably paranoics with systematized delusions, have refused to participate. The majority of the patients enjoy the increased attention and like to attend the group meetings.

Of equal importance is the effect on morale and attitude of the attendants. Their self-esteem is heightened; they have become aware that they are not working in an asylum where merely custodial care is given but in a hospital where every effort is made to get patients well. Several desirable applicants including psychology students have applied for positions as attendants when they learned of this project. An attendant who has participated in group therapy will apply his experience to the rest of his work on the ward. This attitude is not limited to the group leaders but spreads to the other

employees. In this way group therapy helps the personnel to achieve the most important factor in a mental hospital: the therapeutic attitude.

SUMMARY

The purpose of this report is to demonstrate how by the use of nurses and attendants as group leaders a large group psychotherapy program can be conducted by a minimum of trained psychotherapists. The group leaders have to be selected carefully, have to be given proper instruction and, most important, have to be continuously supervised in their work by a psychiatrist with experience in psychotherapy.

Such a project was started at the Medfield State Hospital more than a year ago. The instruction received by the group leaders and the supervision of their work is described in detail. Twelve groups conducted by attendants and two groups conducted by nurses are being carried on at present; each group consists of twelve to fifteen patients.

Our experience with group psychotherapy conducted by nurses and attendants has been extremely satisfactory. The patients utilize the opportunity to talk freely and compare their own problems with those of other patients. They learn to verbalize their anxieties and to get along with other people.

The program, also, has had a beneficial effect on the personnel. They have become aware that the seemingly senseless behavior of many psychotics has a definite meaning. An attendant who has participated in group therapy will apply his experience to the rest of his work. This attitude is not limited to group leaders but spreads to the entire hospital.

REFERENCES

1. S. R. Slavson, Qualifications and Training of Group Therapists, *Mental Hygiene*, Vol. XXXI, 386-396, 1947.
2. Gifford and MacKenzie, J.: A Review of Literature on the Group Treatment of Psychoses, Diseases of Nervous System, Vol. IX, 1924-1948.
3. Klapman, J. W.: "The Practice of Group Therapy," editor S. R. Slavson, *International Universities Press*, New York, 1947.
4. Semrad, E. V.: Psychotherapy of the Psychoses in a State Hospital, *Dis. of Nerv. Syst.*, Vol. IX, 105-111, 1948.
5. Mann, J., and Mann, H.: Organization and Technique of Group Treatment of Psychoses, *Dis. of Nerv. Syst.*, Vol. IX, No. 2, 1948.
6. Malamud, W., and Sands, S. L.: A Revision of the Psychiatric Rating Scale, *Am. J. of Psychiat.*, Vol. 104, No. 4, 1947.

Relief of Myopia By Hypnosis and Eye Training

LESLIE M. LECRON
Los Angeles, California

A survey of the literature reveals that abnormal vision is an almost untouched field of psychological investigation. Harrington[1] has called attention to this situation and, himself an eye specialist, remarks that ophthalmologists have shown almost no interest in the psychosomatic aspects of defective or diseased vision, that specialty being the most backward branch of medicine in this respect.

Freud,[2] in describing the mechanism of organ neuroses, was the first to mention the eyes as sometimes neurotically affected. Fenichel[3] gives a brief discussion of ocular symptoms in hysteria, and also states that myopia and micropsia may be conversion symptoms or organ neuroses. The leading texts on psychosomatic medicine have little to say about the eyes. Vision is entirely unmentioned by Dunbar. Weiss and English[4] devote only a few paragraphs to a discussion of the eyes and abnormal vision. They do mention asthenopia as frequently of psychological origin. Alexander and French[5] ignore the eyes with the exception of describing glaucoma as often being psychogenetic, citing several surveys by others as to this disease. Jeliffe[6] reports a case of functional myopia and offers a discussion of disturbed vision from the psychoanalytic standpoint. A few other papers make brief mention of the eyes as vehicles of neurotic symptoms.

*Read before the Society for Clinical and Experimental Hypnosis.

Vision itself might be said to be psychosomatic, for it is a combination of physiological and psychological processes consisting of sensing, selecting and perceiving. The newborn infant is able to sense but it must learn selection and perception before it can "use" its eyes.

Civilized man uses his eyes in different ways than do animals or primitive peoples, such as for reading and other close work. Some of this is under artificial lighting conditions. Eye strain is usually assigned as the main cause for defective vision. Burtt[7] cites Ferree and Rand as showing in a thorough study that eye strain and ocular fatigue are primarily due to variation in lighting in the visual field. The eye is somewhat phototropic and there is a constant readjustment of the accommodation muscles. Strain may also develop because most of us fail to use our eyes properly. Some people use their eyes for close work throughout their lives without ill effect.

In a psychoanalytic consideration of vision it is to be noted that sight is a sexual stimulant at times, with great erotic connotations. There may be guilt feelings where the eyes have witnessed morally unacceptable scenes. Rejection of upsetting sexual perceptions may result in inhibition of the sense of sight, partially or entirely. Masochistic elements may be present as well as conversion symptoms or organ neurosis. One recalls the biblical injunction of Jesus, "If thy right eye offend thee, pluck it out." The neurotic may follow this metaphoric injunction. It is obvious that a neurotic might well develop various reasons for not wanting to see clearly, not necessarily to the point of blindness but with distortion of vision.

As an indication of how the eyes may be neurotically affected, Erickson[8] conducted an experiment intended to test the validity of hypnotically induced deafness. When deafness had been established, Erickson noted in some of his subjects that their vision, previously normal, had become affected. In different subjects various aberrations of sight developed spontaneously, such as nystagamoid movements of the eye, loss of peripheral vision, ocular fixation, blurring, and loss of depth and distance perception.

Among abnormal eye conditions which might, at times, be psychogenetic the most obvious one is hysterical blindness. Others are myopia, presbyopia, strabismus, amblyopia, asthenopia, glaucoma, amaurosis, angiospastic retinopathy and even cataract (particularly when it occurs in early life).

Near and far sightedness account for the great majority of abnormal eye conditions and the fitting of glasses is mainly concerned with the correction of these two conditions. Statistics are unnecessary as it is apparent how many of us are affected and must wear glasses. One of the surprising results in England when the practice of medicine was socialized was the enormous number of people who rushed to oculists for glasses when these were provided at state expense. As a result, the manufacturers of optical equipment were completely swamped.

Presbyopia or long sightedness is a condition which usually develops in middle-age, the cause being assigned to hardening of the lens of the eye with age, thus affecting the ability to accommodate. Yet many elderly people retain normal vision, despite the supposed hardening of the eye lenses.

Most cases of myopia or near-sightedness develop in childhood, often around the age of puberty, which may be significant. Ophthalmologists have recently stated that the incidence of myopia has increased 1600% within the last decade. The reason usually assigned is that the school training of children causes eyestrain through too much work with the eyes at too early an age, and with poor lighting. School lighting is notoriously poor and is undoubtedly a contributing cause, but attempts to relieve strain, such as use of larger type in schoolbooks, have failed to lessen the incidence of myopia among children.

Myopia and presbyopia are both disturbances of the power to accommodate the eyes to near and far vision. Until the Helmholtz theory was propounded, there was much debate as to the reasons for these two conditions. Helmholtz attributed accommodation to the action of the ciliary muscle upon the lens of the eye and this is the accepted theory today. As early as 1918 Dr. W. H. Bates,[8] a New York ophthalmologist, questioned this theory and stated that the real

reason is distortion of the spherical shape of the eyeball. His hypothesis is that the focusing of the eye on near or distant objects is a result of the lengthening or shortening of the eyeball as a whole. He reasons that distortion of the eyeball becomes more or less permanent because of tension of the eye muscles which squeeze the ball into an egg-shape. The long axis is horizontal in myopia, vertical in presbyopia. As is well-recognized, modern living provokes tension in most of us and this might easily involve the eye muscles. This could include the ciliary muscles and the recti and obliques muscles which move the eyeball.

Dr. Bates' theory has never been accepted in medical circles and is regarded as entirely non-orthodox. No scientific investigation has ever been made of Dr. Bates' claims, either to prove or disprove them. However, teachers of his system of eye training have been able to show beneficial results which can be attested to by literally thousands who have taken the Bates course of instruction. Such testimony does not prove the theory and benefit is undoubtedly partly psychological.

The Bates system has more recently been improved and empirically elaborated upon by the American Association for Eye Training group, which has added a consideration of psychological as well as physiological factors in their teaching. Essentially, these systems aim at relieving muscular tension of the eyes and at instructing in the proper methods of using the eyes. Theoretically, normal vision will return when this is accomplished. The theory and methods are described by Aldous Huxley[9] in his book, "The Art of Seeing," written from his own experience in overcoming near blindness, and by Mrs. Corbett.[10]

It is not the purpose of this paper to debate a controversial matter such as the Bates theory, particularly since the writer is not an authority on ophthalmology nor on the Bates method. However, it has been necessary to explain the Bates teachings in order to prepare a background for the case which is to be cited, because these teachings are involved.

In the book *Hypnotism Today*[11] by Dr. Jean Bordeaux and this writer, mention was made briefly that the use of hypnosis, which brings spontaneously a great degree of relaxation, might effectively augment the Bates method of eye training, since that system relies greatly on teaching relaxation. Shortly after publication of the book, a young man who had read it approached the writer, stating that he was myopic and had taken Bates training over an extended period but without benefit. However, he was still convinced that this was something which could help him and was determined to correct his vision if it were possible to do so. He felt that hypnosis might aid in overcoming eye tension.

So far as is known to the writer, nothing along this line had been attempted, so it was decided to experiment. A Bates teacher, not the one heretofore visited, was enlisted in the test. It was assumed that the best approach would be to teach the young man, whom we will call B——, autohypnosis so that he could receive Bates instruction while in the hypnotic state and hence completely relaxed. With this procedure, the presence of the hypnotist would not be required. Accordingly, Bates lessons were given as usual but with particular stress laid on obtaining muscular relaxation of the eyes through a combination of eye exercises and suggestion, B—— first placing himself in a hypnotic trance.

B—— proved to be a somnambulistic subject, able to enter a deep trance within a few moments, with subsequent amnesia if it was suggested. He soon learned to hypnotize himself and to remain in the trance with eyes open, maintaining mental alertness. The Bates lessons were given three times a week, each one lasting an hour and a half. Details of the Bates techniques are unimportant here and will not be described.

B——, aged 26, was a college graduate of high intelligence. He had taken several courses in psychology and had read on psychotherapy and psychoanalysis. He recognized that he was neurotic but did not believe he was suffering from an actual neurosis since he functioned competently. He faced reality well and accepted responsibility. His neurotic traits were minor and he probably could be correctly diagnosed as having a mild character neurosis. It should

be mentioned that he is a very handsome, personable young man, a point the importance of which will be seen later. At the time of his first visit, his vision had been tested and found to be 20-200 in each eye. He, accordingly, wore corrective glasses. As he expressed it, "I can't see a billboard across the street without them."

Using the technique of hypnotizing himself at the beginning of each lesson, within a few sessions good results were noted. He began to have the flashes of normal vision which are frequently observed in Bates' work. As a matter of fact, it is the endeavor of Bates teachers to prolong these flashes, cause them to appear more and more often, and then eventually to bring back continuous normal vision. B—— soon developed a remarkable control of the eye muscles in regard to relaxing them, but such release of the tension was only temporary.

After two months, B—— informed the writer that he believed psychological factors were involved in his condition—that he was blocking himself from achieving normal vision. He felt that he was making progress, since he experienced normal vision for brief periods, but something interfered with its maintenance. He believed that hypnoanalytic sessions might quickly uncover causes. This was actually accomplished in only two sessions, no effort being made at more than the most superficial analysis, which was mostly ventilation and a discussion of his subconscious motivations. Insight seemed to come at once.

Under hypnosis it was pointed out that there might be definite neurotic motivations for his nearsightedness. He was questioned as to the first time he had noticed any difficulty with his vision. This had begun when he was aged thirteen. He recalled that the condition had developed rapidly within a period of about three months, the myopia progressively increasing with the passage of time. Prior to entering the third year of junior high school, he could not remember having had any trouble with his eyes. When he was thirteen, his mother had left the family home, deserting her husband and three children, brazenly going to live in the same neighborhood with another man. The father had been glad to be rid of her and did nothing about the situation. While the father was indulgent and kind, the mother had never shown affection for the children, had punished them unjustly, and dominated them. B—— stated that he had never been fond of her and had been resentful and rebellious. After she left the home, he found himself hating her deeply and bitterly. This was complicated by the fact that she still lived nearby and he would occasionally meet her on the street, at which times he avoided her by entering a store or crossing the street. He was continually afraid of encountering her. Still under hypnosis, he said, "I never wanted to see the woman again! I couldn't stand the sight of her."

At this remark he sat up and exclaimed, "Why that's a good reason for my having become near-sighted!" There was then a discussion as to the possibility of his sight having been "drawn in" to avoid seeing the mother.

It was further elicited that during this period he was greatly upset emotionally, was unable to study, and his grades were poor where formerly they had been excellent. He hated to recite or to look at the blackboard. His vision began to deteriorate so that his seat in school was moved from the rear of the class to the front, so that he could see the blackboard.

At the next session, while talking of his childhood, B—— said:

> "I was born a real monstrosity in appearance. All through my childhood I became more and more aware of this. I had a huge hooked nose and protruding ears so large they almost flapped when I walked. I was so repulsive that other children avoided me and I had no playmates. They often taunted my ugliness. I was really a terrible sight and I am not exaggerating. When I was older, I hated to shave because I saw myself in the mirror. I always avoided mirrors. I looked so horrible that I never wanted to see myself."

Immediately he saw the significance of these remarks and commented that here was another reason for retraction of his vision.

B—— then explained that his repulsive appearance had led him to undergo plastic surgery. This had been most successful and only by close observation could one see the many tiny scars from the operations. He was now a fine-looking young man. This fact was stressed to him and he agreed that he was no longer concerned about his looks.

B—— stated that he believed the factors now uncovered were the basic causes of his near-sightedness. Under hypnosis strong suggestions were given that he now had an understanding of his condition and no longer needed his symptoms because his appearance was satisfactory and he had made an adjustment to the mother situation after she married again so that he was now friendly with her. He was told his vision could now become normal and that further Bates lessons would quickly break up the habit patterns which had been formed.

Continuation of the lessons brought good results. At the end of six months, total elapsed time, his eyes were nearly normal, so far as he could tell. His glasses had been discarded and at present it has been a year and a half since he has worn them. He was asked to have his vision checked by an ophthalmologist but failed to do so, having gone to work in a desert location where an examination was not feasible.

Discussion

Whether the brief, superficial psychotherapy attempted in this case would have had any effect in relieving the eye condition without the addition of eye training is problematic. It is unlikely that any result would have been accomplished with either hypnosis alone or the Bates work alone. The combination proved successful in a relatively short time. Before psychotherapy was attempted, previous Bates work had brought no benefit. With brief psychotherapy with hypnosis, relaxation of the eye muscles was accomplished and near normal vision returned.

In the course of psychoanalysis, it is suggested that, if the patient wears glasses, the possible psychogenesis of abnormal vision be investigated. It is further suggested that the relationship of myopia to introversion might well be made a subject of investigation. Though it is purely a guess, it is to be suspected that a large percentage of extreme introverts would be found to be near-sighted.

Summary

Consideration is given of the role of subconscious motivation and neurotic factors in disturbances of vision. Tensions affecting the eye muscles are regarded as being a possible cause of abnormal vision. It is considered that hypnosis may be utilized to release such tensions and in connection with the Bates method of eye conditioning, will be of much value in overcoming visual defects. It is pointed out that psychological factors frequently may be involved in myopia and in other eye conditions. A case history is cited of a myope where a relief of the condition was obtained through a combination of brief hypnoanalysis, hypnotic relaxation and Bates eye training.

REFERENCES

1. Harrington, D. O.: Ocular Manifestations of Psychomatic Disorders. *J.A.M.A.*, 133: 669, Mch. 8, 1947.
2. Freud, S.: Collected Papers, Vol. II, p. 110, *Hogarth Press*, London, 1924.
3. Fenichel, O.: Psychoanalytic Theory of Neurosis, *Norton*, New York, 1945.
4. Weiss, E., and English, O. S.: Psychosomatic Medicine, *Saunders*, Philadelphia, 1943.
5. Alexander, F., and French, T. M.: Studies in Psychosomatic Medicine, *Ronald Press*, New York, 1949.
6. Jelliffe, S. E.: Psychoanalysis and Organic Disorder: Myopia as a Paradigm, *Intl. J. Psychoan.*, 7, 1926.
7. Burtt, H.: Psychology and Industrial Efficiency, pp. 229f. *Appleton*, New York, 1931.
8. Erickson, M. H.: Hypnotic Investigation of Psychosomatic Phenomena, *Psychosom. Med.*, V. No. 1: 51, 1943.
9. Bates, W. H.: Perfect Sight Without Glasses, *Henry Holt*, 1918.
10. Huxley, A.: The Art of Seeing, *Harpers*, New York, 1942.
11. Corbett, M. D.: Help Yourself to Better Sight, *Prentice Hall*, New York, 1949.
12. LeCron, L. M., and Bordeaux, J.: Hypnotism Today, *Grune & Stratton*, New York, 1947.

Regressive Shock Therapy In Schizophrenia

DAVID ROTHSCHILD, M.D., DONALD J. VAN GORDON, M.D., and ANTHONY VARJABEDIAN, M.D.

Worcester, Massachusetts

In electro-convulsive therapy of mental disorders it is not uncommon to give a more intensive course than the customary one consisting of three treatments weekly. Such a procedure has been advocated, particularly for acute manic states, by Kalinowsky and Hoch,[1] Kino and Thorpe,[2] and more recently for depressive and paranoid reactions by Valentine.[3] Usually one to three treatments were given daily, with a rapid tapering off to a slower rate of administration.

In recent years, still more intensive procedures have been advocated. According to Cerletti,[4] Bini suggested the repetition of electroshock many times a day for certain patients, naming the method "annihilation." In 1948, Kennedy and Anchel[5] described under the term "Regressive electric-shock" an intensive method for intractable schizophrenic psychoses. Their object was to give electrically induced convulsions with sufficient frequency to produce a state of deep regression. They started by inducing 2 to 4 grand mal seizures daily until the desired degree of regression was reached. Their shortest course consisted of 11 seizures and their longest 50, but generally 20 to 30 were given. They considered that a patient had regressed sufficiently when he wet and soiled himself or acted and talked like a 4 year old child. Their patients were confused and could not take care of their physical needs; they lost weight and frequently had to be spoonfed. The period of posttreatment confusion lasted for about 10 to 14 days.

Kennedy and Anchel[5] reported the results of this form of treatment in 24 patients who had proved refractory to other forms of shock therapy. Eight of their patients were able to leave the hospital, 6 were described as in partial remission but still in the hospital because of a lack of suitable facilities for home placement, 11 others were regarded as improved and only 1 patient failed to show any improvement.

In view of these highly favorable results, a similar intensive course of electric-shock therapy, somewhat modified as described below in an effort to standardize the procedure, was administered to 52 schizophrenic patients at the Worcester State Hospital.

Material

The patients were selected on the basis of failure to show lasting improvement with other methods of therapy. Since it was desired to explore the potentialities of the method, the choice of cases was not restricted to those showing some of the more favorable prognostic features, such as acuteness of onset or preservation of affect, and the like. There were 27 male and 25 female patients in the group. All patients had received ordinary courses of electric shock or metrazol therapy with the exception of 1 patient who had been given only a course of 50 insulin coma treatments. In addition to the convulsive therapy, 8 patients had been treated with insulin coma and 4 with insulin subcoma. Most of the patients had in fact received two or more courses of shock therapy.

Because of the intensive nature of the treatment the choice of cases was limited to relatively young persons in good physical condition. The ages of the male patients ranged from 21 to 51, with an average of 33.5 years, and those of the female patients from 20 to 42, with an average of 32.8 years.

The duration of the illness, dating from the first attacks, for the male patients varied from 13 months to 22 years, with an average of 8.8 years and a mean of 8.1 years, and for the female patients from 1 to 15 years, with an average of 5.7 years and a mean of 5 years. Counting only the present attack, the duration of the illness for the male patients ranged from 9 months to 22

From the clinical services of the Worcester State Hospital. Read at a meeting of the Massachusetts Society for Research in Psychiatry held at the Cushing Veterans Administration Hospital, Framingham, Mass.

years with an average of 7.1 years and a mean of 4.5 years, and for the female patients the range was from 7 months to 15 years with an average of 3.8 years and mean of 2.2 years.

Method

It was decided to standardize the method by giving 4 grand mal seizures daily, spaced so that 2 were given in the morning at one to two hour intervals and 2 in the afternoon, for 7 consecutive days, the aim being to produce a total of 28 major seizures in each case. This aim could not always be accomplished, for certain patients built up such a tolerance that they sometimes had petit mal attacks and major convulsions could not be produced without exceeding what was considered to be a safe dosage. Thus, of the 27 male patients, 1 received only 14 major seizures and another 17; 4 patients sustained 21 and 7 patients sustained from 22 to 27 grand mal attacks. All the male patients were able to finish the course of treatment. Among the female patients, 1 was discontinued from treatment after 12 major seizures because of marked resistiveness, refusal to eat or drink and consequent dehydration. Another was discontinued after 20 seizures because of refusal to eat or drink, and in a third case treatment was stopped after 23 seizures, when the patient developed a rapid and irregular heart rate which lasted for 18 hours. In a fourth case the treatment was stopped after 24 seizures because the patient appeared to be in a state of prostration and collapse. This patient recovered after a few days' rest in bed with no untoward results.

By the end of this intensive course of treatment practically all patients showed profound disturbances. They were dazed, out of contact and for the most part helpless. All showed incontinence of urine, and incontinence of feces was not uncommon. Most of them were underactive and did not talk spontaneously. Many failed to respond to questions but a few patients would obey simple requests. They appeared prostrated and apathetic. At the same time most of them whined, whimpered and cried readily, and some were resistive and petulant in a childish way. They could usually be made to walk if led and supported, but their movements were slow, uncertain and clumsy. Most of them liked to be coddled. Masturbation was not uncommon. They seemed to have lost all desire to eat or drink and showed no discrimination as to what they were eating. They had to be spoonfed, and most of them lost from 3 to 12 pounds in weight during the course of treatment. They could not dress themselves and none of those tested during this period could complete the task of extracting a match from a match box and lighting the match.

The foregoing symptoms usually began to appear during the course of treatment, but became fully developed at the end of the course and lasted for approximately one to two weeks thereafter. Recovery was gradual, with the ability to walk, to feed oneself and to talk ordinarily being re-established within a week after termination of treatment. Incontinence usually ceased at about this same time. Interest in their own appearance and in persons or objects was shown as a rule for the first time during the second post-shock week.

Our procedure was to treat from 5 to 7 patients at one time, with a special nurse and 2 student nurses assigned to the group during the hours of treatment. This nurse continued to care for the patients for several weeks after the termination of treatment and was the leader in a planned program of rehabilitation in which habit training, occupational therapy and recreational activities were stressed. It is our impression that this program of rehabilitation was an important factor in producing improvement.

Results

The results at the end of a follow-up period ranging from 3 to 15 months are presented in Table 1. Of the total group, 13 patients have been out of the hospital for periods varying from 3 to 12 months. In addition to these patients, 5 patients had been home on visit for periods of from 2 to 9 months, but they were returned to the hospital because of a recurrence of symptoms. Among the patients remaining in the hospital, 6 are able to go home on week-end visits and 5 are on open wards.

TABLE 1

RESULTS OF REGRESSIVE SHOCK THERAPY IN 52 CASES OF SCHIZOPHRENIA

Type of Schizophrenia	Number of Cases	Discharged on Visit	Much Improved	Improved	Slightly improved	Unimproved
Catatonic	10	3	1	2	2	5
Hebephrenic	12	2	2	3	1	6
Paranoid	11	4	1	4	2	4
Other Types	14	3	1	3	5	5
Simple	5	1	0	2	1	2
Total	52	13	5	14	11	22

With respect to the degree of improvement noted, 5 patients were regarded as much improved. This term is used for those patients making a good social and occupational adjustment outside the hospital under conditions approximating their prepsychotic level. However, they showed slight residuals, such as poor judgment, shallow or loose thinking, or mild disturbances of affect and associations. Fourteen patients were regarded as improved; they were making a decidedly better adjustment, functioning reasonably well, some of them outside the hospital, though still showing psychotic symptoms and requiring supervision. Eleven patients were slightly improved in the sense that some of their symptoms were less pronounced and their adjustment to the routine of hospital life was somewhat better. Twenty-two patients were unimproved.

A scrutiny of Table 1 indicates that the results were slightly more favorable in the paranoid form than in other types of schizophrenia, but the number of cases was too small to render this finding significant.

As might be expected, the best results were obtained in patients with illnesses of relatively short duration. This is brought out in Table 2. Thus, none of the patients whose present attack of illness had lasted longer than 5 years was out of the hospital, though a few of these patients showed worth-while improvement in their hospital adjustment. Also, a better prognosis was noted in patients who had had earlier attacks with good or partial remissions; there were 13 patients in this category and 6 of them were out of the hospital.

TABLE 2

RESULTS OF REGRESSIVE SHOCK THERAPY IN SCHIZOPHRENIA ACCORDING TO DURATION OF ILLNESS

Duration of Illness (Present Attack)	Total Number of Cases	Discharged on Visit	Much Improved	Improved	Slightly improved	Unimproved
Under 3 years	22	9	5	5	2	10
3-5 years	12	4	0	5	2	5
5-10 years	11	0	0	3	5	3
10 years	7	0	0	1	2	4

Comment

The foregoing results are less favorable than those of Kennedy and Anchel[5] but more favorable than those recently reported by Weil,[6] who used regressive electroplexy in 18 patients with no lasting beneficial effect. In 36 per cent of our patients considerable improvement occurred and in 21 per cent slight improvement was noted. In 25 per cent of the total group return to the community was accomplished. Five other patients had to be brought back to the hospital after they had been at home for from 2 to 9 months. This suggests the likelihood that with a longer follow-up period, a recrudescence of symptoms may occur in some of the patients who are now adjusting well. Even so, the results are encouraging because they indicate that a certain number of patients who are intractable to customary methods of treatment and who appear to be advanced schizophrenics still possess surprising potentialities for improvement. The fact that this improvement may be only temporary should stimulate further research in the field of therapy for such patients.

The question arises whether regressive shock therapy may produce permanent cerebral damage. Our patients did not show any significant neurologic changes, and clinical psychiatric observations did not reveal any organic mental impairment apart from the temporary changes already described. More detailed psychologic studies are being performed, but as yet the number of patients tested is too small to warrant any conclusions. Electroencephalograms were taken in many of the cases before, during and after the course of treatment. The observations have been reported by Callaway,[7]

who found certain unusual changes in slow wave activity of a temporary nature but no evidence of permanent alterations.

One may wonder whether the state of confusion and prostration produced by this method of treatment should be called "regressive" in the sense that it conforms to the psychopathologic concept of regression, particularly as developed in psychoanalytic theory. In this connection the following observations may be of interest. Toward the end of a course of treatment, one patient seemed totally unable to swallow and since she was taking no fluids whatsoever, treatment was discontinued after 20 seizures had been given. The neurologic examination was negative. When fluid was poured into her mouth, it ran out directly or accumulated in the bottom of her mouth and flowed out later. However, when a standard nursing bottle with nipple was placed in her mouth, she began to suck and was able to swallow the milk slowly but completely. To our further surprise, it was found that the other patients being treated at this time also readily fed from nursing bottles, and they continued to do so for 9 days after the cessation of treatment. It would thus appear that the term "regressive" is justified in our cases in so far as it designates a return to an early, infantile mode of activity in one specific sphere.

Cerquetelli and Catalano[8] studied the changes associated with intensive electroshock therapy in terms of an "annihilation syndrome." They compared it with the psychopathology following prefrontal leukotomy and indicated a close parallelism with the advantage of reversibility in the case of shock.

In conclusion, it should be emphasized that regressive shock therapy is a drastic procedure which requires close medical supervision and intensive nursing care. It is justified only in cases where standard methods of treatment have failed and the course of the illness indicates such a poor prognosis that the final step of lobotomy may be considered. Our observations suggest that regressive shock therapy may be successful in salvaging a small but worth-while proportion of such cases.

Summary

An intensive method of electro-convulsive therapy is described under the term regressive shock therapy. Four treatments are given daily at spaced intervals for seven consecutive days.

This form of treatment was administered to 52 schizophrenic patients who had failed to show lasting improvement with more conservative forms of shock therapy.

Five patients were much improved, 14 were improved, 11 were slightly improved and 22 were unimproved at the end of a follow-up period ranging from 3 to 15 months. Thirteen patients, or 25 per cent of the whole group, have been out of the hospital for periods varying from 3 to 12 months.

Although no permanent ill effects have been noted, regressive shock therapy is regarded as a drastic procedure which requires close medical supervision and intensive nursing care. It is justified only in cases where standard methods of treatment have failed and the course of the illness indicates a poor prognosis.

BIBLIOGRAPHY

1. Kalinowsky, L. B., and Hoch, P. H.: Shock Treatment and Other Somatic Procedures in Psychiatry. New York, *Grune and Stratton,* 1946.
2. Kino, F. F., and Thorpe, F. T.: Electrical Convulsion Therapy in 500 Selected Psychotics. *J. Ment. Science,* 92: 138, 1946.
3. Valentine, M.: Intensive Electroplexy. *J. Nerv. Ment. Dis.,* 109: 95, 1949.
4. Cerletti, U.: Old and New Information About Electroshock. *Am. J. Psychiat.,* 107: 87, August, 1950.
5. Kennedy, C., and Anchel, D.: Regressive Electric-Shock in Schizophrenics Refractory to Other Shock Therapies. *Psychiat. Quart.,* 22: 317, 1948.
6. Weil, P. L.: "Regressive" Electroplexy in Schizophrenics. *J. Ment. Science,* 96: 514, 1950.
7. Callaway, E.: Slow Wave Phenomena in Intensive Electroshock. *EEG Clin. Neurophysiol.,* 2: 157, 1950.
8. Cerquetelli and Catalano. Quoted by Cerletti. *Ibid.*

Neurological Clinical Pathological Conference

of the

Cincinnati General Hospital

CHARLES D. ARING, M.D., *Editor*

Case No. 263443

Presentation

A 69-year-old white woman had been well except for a slight cough and mild exertional dyspnea until about one year before admission to the hospital. In mid-April 1950 she began to cough blood-tinged sputum. Two weeks later she developed a catching pain in the chest. On May 8 pain and swelling of the left leg appeared.

Physical examination on May 9, at the time of hospital admission, demonstrated a blood pressure of 130/80 and a sinus tachycardia of 124 per minute. There were physical signs suggestive of pleural fluid at the base of the left lung. The left leg was swollen and there was tenderness of the calf muscles and behind the knee. Homan's sign was present on the left (pain in the calf when the foot was forcibly dorsiflexed). There was slight edema of the right leg. A roentgenogram of the chest showed considerable enlargement of the heart in its transverse diameter and a small pleural effusion at the left base. An electrocardiogram showed evidence of left ventricular strain. On May 10, 280 cubic centimeters of blood-tinged fluid was removed from the left pleural space.

Treatment was begun with heparin and dicumarol on the day of admission and the prothrombin time of the blood fell progressively from an initial value of 78 per cent to a low of 10.8 per cent on May 14. The prothrombin time did not rise above 17.6 per cent until after dicumarol was discontinued on May 23. Sinus tachycardia was persistent throughout the illness and on May 17 a paroxysm of auricular tachycardia was demonstrated by electrocardiogram.

On May 17 the patient first complained of stiffness of her arms and fingers. She was unable to turn in bed. She had felt chilled all night and became incontinent of urine. On the following day weakness of all four extremities was demonstrable and the deep reflexes which were normal on admission had become hypoactive. Within the next two days flaccid paralysis of all extremities developed and the deep reflexes disappeared. The patient became unable to raise her head but could turn it from side to side. On May 22 examination of the cerebrospinal fluid showed an initial pressure of 88 millimeters of water; 5 cubic centimeters of clear fluid were removed and the final pressure was 35 millimeters. The fluid contained 143 milligrams per cent of protein and no cells. The spinal fluid Wassermann test was negative and the colloidal gold reaction was normal. Manometric studies showed no evidence of subarachnoid block.

Later neurological examinations showed a return of only slight movements of the fingers and weak radial and biceps reflexes. There was no improvement in muscular activity after the first week in June. The cerebrospinal fluid protein was 188 milligrams per cent on May 28, 130 on June 12, 120 on August 4, and 65 on August 28. The patient remained incontinent of urine and became incontinent of stool. Decubitus ulcers developed and there was a gradual fall of plasma albumin. Antibiotics and a retention catheter were used at times in attempts to promote healing of the decubiti but a cystitis developed which was rather resistant to treatment. On August 27 the blood pressure fell to shock level and the patient became comatose. The temperature which had been relatively normal throughout the illness rose to 105.4 degrees Fahrenheit and innumerable petechiae appeared over the trunk. There was little response to intensive antibiotic and supportive therapy and the patient expired on September 1.

Differential Diagnosis

Dr. Richard Vilter: This patient, a 69 year old white woman, had been well except for a slight cough and mild exertional dyspnea until about one year before admission. These manifestations might indicate that she either had chronic lung disease for some time or, more likely, that she had mild congestive heart failure for a year or more. Then we come to the symptoms that brought her to the hospital. These need some interpretation. She began to spit blood and after two weeks there was pain in the chest and then in the left leg, which began to swell. These symptoms may be interpreted in two ways, the most obvious is that she had a silent thrombosis in the calf of the left leg without symptoms, that an embolus became detached from that thrombosis, that several embolizations probably occurred, accounting for the bloody expectoration and finally for the pain in her chest. This thrombosis eventually mounted and produced certain clinical manifestations after the embolization occurred.

On the other hand, one might reason in another direction and consider that perhaps she had either a tumor such as bronchogenic carcinoma or some serious infection, per-

haps tuberculosis or wide-spread bronchiectasis in her lung, either of which might have lead to the pain and bloody expectoration, and then, because of the tumor or infection, she developed phlebothrombosis in the leg. Tumors, particularly carcinoma of the pancreas, are prone to induce thromboses in veins at distant sites. Any tumor may do it and sometimes infection may also.

I would like to digress briefly from the protocol and elaborate on thromboses in veins. Too little is known about this subject. We do find several classifications. We speak of thrombophlebitis and by that we mean an inflammatory process in veins with heat, redness, swelling, and usually exquisite tenderness which may be localized either over a vein segment or may involve all the veins in the extremities. Such an inflammatory process may be the result of injury, of severe infection as ruptured appendix with generalized peritonitis, or it may occur as a migrating phenomenon secondary to Buerger's disease. We speak about phlebothrombosis, a silent clot formation in a vein with little inflammation, such as may occur after a surgical operation, after prolonged bed rest, in the course of various lymphomas such as Hodgkin's disease, lymphosarcomas, leukemia, etc., and, as previously mentioned, in the course of certain tumors such as pancreatic carcinoma. This is the most dangerous form of blood clot in veins. The clot is friable and breaks up easily and may cause embolization before the primary site makes itself known. Between these two categories, there are many types of thrombotic syndromes, all of obscure etiology. There are various forms of migrating thromboses and thrombophlebitis unrelated to Buerger's disease and, as far as we know, unrelated to anything else except to certain foci of infection about the body, sometimes in the prostate, sometimes in teeth, to mention two possible sites. There is no certainty, however, that these foci have anything to do with the thrombosis.

There is a question whether allergy plays any part in etiology. It has been shown that one can sensitize rabbits to venous endothelium and by such sensitization, followed by an injection of antigen, provoke venous thrombosis and hemorrhage. I mention this more to demonstrate how little we know about vein disease than to give an erudite dissertation on the subject.

I think we can say that this patient had a thrombosis in the deep veins of the left calf. The physical findings corroborate the symptoms. She had a swollen leg, a tender calf, and a Homan sign, that is, when the foot was dorsiflexed, pain occurred in the calf due to stretch in the muscle around the involved vein. She had bloody pleural effusion which we would expect in a person who has had at least several pulmonary infarcts. Since the X-rays showed no tuberculosis, no tumor, no evidence of infection, just fluid in the chest, we can eliminate all these possibilities and conclude that this patient had a silent thrombosis in the veins of her calf, that emboli were detached and lodged in her lungs, and that she had pulmonary infarcts secondary to embolization.

She came to the hospital and was treated in the usual fashion with heparin and dicumarol. The prothrombin concentration came down following dicumarol administration. Then heparin was stopped. The prothrombin concentrations never fell to dangerously low levels but were maintained somewhere between 10 and 20 per cent without difficulty. Everything seemed to be going along well except for persistent tachycardia. That might easily have been explained on admission because of the pulmonary infarct but after 8 days it should have subsided. Nevertheless, it continued and at that point she began to develop stiffness and weakness of the hands and arms which progressed over the course of several days to quadriplegia associated with bladder and rectal sphincter involvement with few sensory manifestations. A spinal tap was done and the pressures were found normal. There were no cells but the protein was elevated. The "albumino-cytologic dissociation" immediately raises the possibility of some type of neuritis, the Guillain-Barré syndrome in which facial palsy is a common manifestation but not a necessary one. Although it seems to me that that would be the best explanation for the course of neurologic events, there are other things which we must consider.

This woman had already had thromboses in veins with embolization. In addition, she

was on anticoagulants. So, one must consider not only additional thromboses in her central nervous system, embolization perhaps to the spinal cord, but also central nervous system bleeding from the anticoagulants. One certainly can have repeated thromboses in the face of adequate anticoagulant therapy. I have seen patients on anticoagulant therapy because of pulmonary infarction and thrombophlebitis who, during the course of such therapy, which was well maintained and well controlled, suffer an acute myocardial infarct due to coronary thrombosis. Other thromboses have occurred also and have been reported during anticoagulant therapy so that the fact that she had this type of therapy does not eliminate the possibility of additional thromboses. The most likely vascular lesions which might have occurred in this patient are as follows: An anterior spinal artery thrombosis or embolization at the cervical level which should have come on suddenly and not over a period of 3 or 4 days. The anterior spinal artery supplies the anterior portion of the spinal cord, mostly gray matter; and also the pyramidal tracts. Therefore, I would have expected extensor plantar responses after the period of shock had passed. There is nothing in the protocol that indicates pyramidal tract involvement.

The patient might have bled into the spinal cord. She might have developed an hematomyelia during anticoagulant therapy as has occurred in patients with hemophilia or thrombocytopenia. The hematomyelia would have had to involve the cervical level in order to explain the quadriplegia. It should have been sudden in onset, much more sudden than the 4 or 5 day progression of events described here and I would have expected some sensory manifestations, possibly the same type one finds in syringomyelia, with dissociation of pain and temperature from touch.

Other conditions to be considered are transverse myelitis or extradural, subdural, or subarachnoid bleeding due to the anticoagulants. Under such circumstances, if bleeding were the cause of her symptoms, I would have expected bleeding elsewhere, some other manifestations of hemorrhagic disease. This she did not have. If she had had epidural or subdural bleeding, I would have expected root pains. I would have expected manifestations of cord compression but she had none. So I think we can eliminate these possibilities. I should point out that a paravertebral block in the face of anticoagulant therapy is contra-indicated because it may lead to bleeding around the cord. If one has a patient with thrombophlebitis on anticoagulant therapy, it would be unwise to do a paravertebral block in an attempt to relieve pain.

Other conditions such as poliomyelitis may be considered. The symmetry of the quadriplegia and the absence of cells in the cerebrospinal fluid rules this out. Post-rabies innoculation encephalomyelitis should be considered because the symptoms and signs may be the same. She had not been exposed to rabies vaccine, so I believe we can eliminate this possibility. One cannot on the physical findings or on the cerebrospinal fluid findings eliminate post-diphtheritic neuropathy. The nervous system manifestations following diphtheria may be exactly the same as were observed in this patient. However, this patient did not have a focus either in the throat or in ulcers of the legs from which diphtheria toxin might have been absorbed. Other even less likely causes of quadriplegia, tic paralysis or acute intermittent porphyria can be rejected for lack of confirmatory evidence. Potassium deficiency, acquired in the course of treatment for diabetic acidosis or during the course of a potassium-losing nephritis, during ACTH or cortisone administration, or on a congenital basis in familial periodic paralysis, certainly can be eliminated in this patient. They should be kept in mind as causes for sudden onset of weakness and flaccidity. Heavy metal poisonings or beriberi peripheral neuropathy can be eliminated since there is no supporting evidence.

These considerations leave me with the Guillain-Barré syndrome as the most logical choice. What is this syndrome? It has been called by a multitude of names. I believe it was back in the 1850's that Landry first described an ascending paralysis which has since borne his name. Since that time there has been argument as to whether this syndrome differed from what later was

called Guillain-Barré syndrome, infectious polyneuritis, infectious neuronitis, polyneuritis with facial diplegia, acute disseminated encephalomyelitis, and other names. I think the evidence at the present time is in favor of the unity of these syndromes. They appear to be the result of some type of toxic degeneration in various parts of the nervous system, particularly in roots and peripheral nerves. There are also changes in other organs and viscera as Sabin and Aring[1] showed in their study on this subject. It may be that various toxic agents lead to this particular type of degeneration through some type of hypersensitivity reaction. Nobody has really defined the exact mechanism.

This syndrome may occur after a mild upper respiratory infection. It also occurs during recovery from diabetic acidosis and in patients who had been receiving sufanomides, also with infectious mononucleosis, infectious hepatitis, and it has been described in the course of serum sickness. As a matter of fact, one of the laboratory tests which is sometimes positive in Guillain-Barré syndrome is the heterophile agglutination. It has occurred during the course of pregnancy and in the puerperium, during the course of syphilitic infection, and after exposure to mustard gas. So you see there are any number of predisposing factors. I have already indicated that diphtheria can produce a similar syndrome.

The patients who have the Guillain-Barré syndrome may get over the disease in the course of several weeks or it may continue for months and gradually improve. A chronic phase may develop with cord bladder and ascending urinary tract infections and bed sores lasting months and even years. Finally death may result as this patient died in terminal sepsis. A chronic recurring form has been described; it must be extremely rare.

Finally, there is a question in my mind as to whether dicumarol may have had something to do with our patient's neuritis. I looked through the literature to find another case of Guillain-Barré syndrome that followed dicumarol ingestion without success.

This patient died of sepsis arising either from bed sores or from urinary tract infection; it is impossible to say which. The purpura was a manifestation of damage to capillaries by the systemic infection.

In summary, this patient had a phlebothrombosis in the left calf, silent at first, with emboli to the lung. During the course in the hospital she developed the Guillain-Barré syndrome. I use that term because of the non-specificity of the reaction and because I don't believe this is an infectious process per se, although it has been termed infectious neuritis. She had mild arteriosclerotic heart disease. She died of terminal septicemia.

Dr. Howard D. McIntyre: I agree with Doctor Vilter's concept of the Guillain-Barré syndrome as a non-specific reaction except in one group of cases. These begin with upper respiratory infection which clears within a week or ten days, and the patient is apparently well when the neurological symptoms appear. These patients may improve and recover within a few weeks, or they may die of an ascending paralysis, or from heart involvement just as in diphtheria.

Facial palsy is not present in all cases of the Guillain-Barré syndrome; it may be found in diabetic neuritis, alcoholic neuritis, etc. Facial palsy depends on the severity of the swelling of the seventh nerve. Since it traverses a long, boney canal, a modicum of swelling of the nerve results in facial palsy for mechanical reasons. I don't think facial palsy adds any specificity as far as the syndrome is concerned.

The distinction between proximal and distal muscle weakness has been stressed but I don't believe that it is of importance. I have seen the classical syndrome begin peripherally, I've seen it begin proximally, and the weakness is usually as severe in one portion of an involved limb as another.

Dr. Milton Rosenbaum: What about nerve tenderness?

Doctor McIntyre: It may or may not be present; some cases are purely motor.

Dr. Warner Peck (demonstrating the roentgenograms): These are representative films from a series, the last taken on June 17. On admission, the transverse diameter of the heart was greatly increased over that

normally expected for a patient with this chest size. At the left base there was fluid. At the right base there was a little clouding —I can't say whether there was fluid there or not. None was found on physical examination at that time.

The presence of fluid in the left chest of a patient where the heart is large, and cardiac failure is suspected clinically, suggests the possibility of infarction on the left because the majority of pleural effusions with heart failure occur on the right side. When they occur on the left side in the presence of heart failure and no infarction, it is usually the result of obliterative pleurisy at the right base, making it impossible for any fluid to collect in this area. This last film, taken approximately six weeks after the first one shows a triangular shaped infiltrate just superior to the minor fissure on the right and probably represents an infarction in the right hemithorax at that time. The patient, therefore, was probably suffering from multiple pulmonary infarctions as Doctor Vilter suggested.

Skull films made in the middle of her hospital stay are not remarkable except for the presence of hyperostosis along the internal surface of the frontal bone. Some individuals term this condition metabolic craniopathy. At one time Doctor Sherwood Moore of St. Louis felt that a significant number of these patients were suffering from metabolic and endocrine disturbances and that the hyperostosis of the inner table was a result of disease. However, Doctor Wisoff of the X-ray Department took skull plates of fifty persons in order as they came through the Receiving Ward. He found an incidence of hyperostosis frontalis interna of 5 per cent which is the same percentage that Doctor Moore noted in sanitarium patients. We feel that the condition has no known clinical significance.

Pathological Discussion

Dr. Murton R. Shaver: Necropsy was performed 4 hours post mortem. Abnormalities outside of the nervous system were: Thrombosis of left popliteal, femoral and common iliac veins; pulmonary embolus, right upper lobe; pulmonary infarction, right upper lobe (old and recent); bilateral hydrothorax; diffuse lobular pneumonia with partial atelectasis of lower lobes bilaterally; and cardiac hypertrophy.

The brain weighed 1200 grams and was removed in continuity with the spinal cord. Superficial inspection revealed no significant abnormalities except for slight widening of the sulci over the superior cerebral surfaces with slightly thickened leptomeninges. The vessels were markedly congested, and the circle of Willis showed occasional calcified plaques in the larger vessels, which were otherwise not remarkable. Serial, coronal sections made through the cerebrum and brain stem and cerebellum failed to reveal any significant abnormalities aside from small punctate hemorrhages, a few of which were found in the region of the third ventricle and in the dentate nuclei, and in the pons and medulla in the region of the fourth ventricle.

Superficial examination of the spinal cord revealed some congestion of the leptomeningeal vessels. Serial sections made through the cord demontrated small disseminated hemorrhages in the gray matter, particularly in the anterior horn regions, in the lower cervical region, and in the lumbar region of the spinal cord. No change in consistency or appearance was noted in the white matter.

Tissue was removed from the cerebral cortex, basal ganglia, midbrain, pons, medulla, and cerebellum and from several levels in the spinal cord and stained with hematoxylin and eosin, phosphotungstic acid, and according to the methods of Loyez and Bielschowsky.

Sections through the cerebral cortex were not particularly remarkable aside from a slight patchy nerve cell loss noted particularly in the third layer of the cortex, while the capillaries and smaller venules generally showed some fibrous thickening of their walls. Around a few of the vessels there appeared to be slight rarefaction and in a few cases small perivascular hemorrhages. There was no evidence of inflammatory infiltration or glial proliferation.

Sections through the basal ganglia and cerebral peduncle disclosed a number of medium and small perivascular hemorrhages scattered in the substantia nigra and ad-

jacent internal capsule with a few in the inferior portion of the wall of the third ventricle. There was no inflammatory infiltration or evidence of gliosis in these regions. The nerve cells in the optic thalamus showed chromatolysis and swelling, while the blood vessels generally appeared congested. Perivascular rarefaction was a little more marked in some areas here, but again it was not associated with cellular infiltration or glial proliferation. The ependyma of the third ventricle appeared normal.

Sections through the pons and cerebellum exhibited marked loss in nerve cells, both in the dentate nucleus and of the Purkinje layer. The cells remaining showed chromatolysis, marked swelling, and severe degenerative changes. In these areas the endothelial cells of small capillaries stood out prominently, but did not appear to be increased in number. The tegmentum nuclei of the pons showed swelling and chromatolysis of many of the nerve cells with a few showing severe degenerative changes. Scattered in the floor of the third ventricle and throughout the tegmentum were small perivascular diapedesis and rarely around one of these vessels there was a slight accumulation of lymphocytes. Some of the larger vessel walls showed swelling of the endothelial cells with loss of nuclear staining and early necrosis.

Sections through the medulla showed numerous small perivascular hemorrhages scattered in the region of the floor of the fourth ventricle and to a lesser extent in areas away from this. In this region the nerve cells showed severe degenerative changes with loss of nuclei and structural outline in many cases, and there was marked diminution in the number of cells present. Around a few of the small capillaries and venules there were small collections of lymphocytes and there appeared to be diffuse generalized slight increase in endothelial cells. A number of the nerve cells in the dorsal vagal nuclei appeared to be well preserved.

Sections made through the spinal cord at the level of the lower cervical, mid-dorsal, lumbar, and sacral levels disclosed disseminated areas of small to medium sized perivascular hemorrhage confined to the gray matter of the cord and most marked in the dorsal and cervical levels. A few of these were also present in the lumbar region, but none were seen in the sacral levels. In the lower levels areas of rarefaction were noted in the anterior horns, whereas hemorrhages had been most marked in the upper levels. Through the cord there was a diffuse increase in endothelial cells in the gray matter.

Many of the smaller vessel walls showed marked thickening and stained poorly. The nerve cells generally showed marked swelling and chromatolysis, some with loss of nuclei, and the most severe changes are seen in the anterior horn cells and lateral horn cells. In places there was sparing of the nerve cells in the lateral anterior group. Many of the nerve cells in Clarke's column showed degenerative changes, with swelling, loss of nucleus, and occasionally vacuolization. The white matter generally throughout the cord was well preserved. The spinal roots in the cervical and dorsal levels showed diffuse swelling of myelin sheaths with vacuolization, tortuosity, and occasionally complete destruction. There was no inflammatory infiltration and very little fibroblastic proliferation. In the lumbar roots and the cauda equina the myelin sheaths were lost in many areas and diminished in number in many of the rootlets. There was diffuse increase in cellularity with proliferation of fibroblasts, but no evidence of lymphocytic or any inflammatory cell infiltration. The neurofibrils showed varying degrees of swelling and fragmentation or loss.

Sections of peripheral nerves showed an appearance somewhat similar to that described in the spinal roots with more widespread replacement of the neurofibrils and myelin sheaths by connective tissue. Rare small lymphocytic aggregations were seen.

Sections of muscle taken from lower extremities showed marked destruction of the muscle fibers with loss of cross striation and in many areas complete replacement by connective tissue. There was some proliferation of the sarcolemmal nuclei, but no evidence of inflammatory cell infiltration.

Clinical Diagnosis

Infectious polyneuritis.

Thrombophlebitis of left leg.

Pulmonary infarction.
Lobular pneumonia.
Terminal septicemia.

Doctor Vilter's Diagnosis

Infectious polyneuritis.
Thrombophlebitis of left leg.
Emboli to the lung.
Arteriosclerotic heart disease.
Terminal septicemia.

Anatomical Diagnosis

Infectious polyneuritis.
Hemorrhagic polio-encephalo-myelopathy.
Thrombosis of the great veins of the right lower extremity and pelvis and left popliteal and femoral and common iliac veins.
Pulmonary embolism with old and recent infarction of right upper lobe.
Bilateral hydrothorax.
Cardiac hypertrophy.

Doctor McIntyre: Are hemorrhages in the spinal cord a part of the picture of infectious polyneuritis?

Doctor Shaver: They have been rarely found in the white matter.

Doctor Vilter: It was noted in the protocol that on August 27 the blood pressure fell to shock levels. The patient became comatose and the temperature rose to 105° F. Petechiae appeared in the skin, and the patient died on September 1. With the hemorrhages in the skin, which suggest terminal sepsis, and the rapid rise in temperature and fall in blood pressure, may not what you see in the cord be a reaction to sepsis without tissue response?

Dr. Charles D. Aring: It would be a most unusual reaction. I can't recall ever having seen hemorrhage limited to gray matter associated with septic death, and many of our patients have died in the manner you describe.

Dr. Helen Glueck: A problem to be considered in this case is whether or not the hemorrhages in the spinal cord were due to dicumarol. Doctor Ovid Meyer has discussed the experimental lesions produced in animals given lethal doses of dicumarol. The characteristic pathologic lesion consisted of marked capillary dilatation and diapedesis of red cells through capillaries. The vascular walls, however, were intact. The dilatation appeared widespread in almost every organ. I wondered if other organs in this patient showed this characteristic pathologic picture.

This raises the problem of anticoagulant therapy in brain disorders, a problem which, I think, is still unsettled and has obvious clinical implications.

Doctor Shaver: That was gone into very thoroughly, and only small hemorrhages in the capsule of the spleen and a few in the lung were found.

Doctor Glueck: Was there a diapedesis of red cells?

Doctor Shaver: No. Congestion of the small venules and capillaries is a common occurrence in the central nervous system.

Dr. Samuel Trufant: Can any comment be made as to the age of the hemorrhages in the cord?

Doctor Shaver: No, as I already mentioned, I can't date them except that there is no evidence of reaction to them.

Doctor Aring: The patient had no anticoagulants for three months before her death.

Doctor Shaver: I would think the lesions more recent than that.

Dr. Gene L. Usdin: Would a ligation of veins have been contemplated early especially if she had been younger?

Doctor Vilter: I believe that any patient who continues to show evidence of embolism coming from a known area, under good anticoagulant therapy, would be considered for vein ligation after the anticoagulant was discontinued. We treat a patient a lot longer with anticoagulant therapy before we ligate veins, and we certainly don't ligate veins before we try anticoagulant therapy.

Student: Isn't polyneuritis characterized by pain? I was wondering why this case comes within the classification of polyneuritis when there was no pain.

Doctor Aring: Polyneuritis usually is characterized by pain and tenderness. However, some few cases do not suffer pain and the typical picture so depicted in the textbooks is not often matched. It seems to be characteristic of students and young physicians to expect the syndrome, whatever it be, to be complete. As far as I know, the textbook never quite catches the human being, and this feature particularly is what makes medical practice so interesting.

Student: I was wondering about rectal and bladder difficulties in infectious polyneuritis.

Doctor McIntyre: I believe that bladder disturbance is an evidence of peripheral neuritis in this case and not evidence of cord disease. I believe that there exists weakness of external sphincters which is a voluntary affair and a neuritic symptom. It is not too common in the milder forms of infectious polyneuritis, and it is present in the severer forms, but it is not a prominent symptom in the majority of cases.

Doctor Shaver: I would like to point out that we have had another case diagnosed clinically as infectious polyneuritis with typical history and clinical signs presenting identical findings histologically with this case, with hemorrhages and general changes in nerve cells. He was not subjected to anticoagulant therapy and died within two weeks of the onset.

REFERENCE

1. Sabin, A. B., and Aring, C. D.: Visceral Lesions in Infectious Polyneuritis. *Amer. J. Path.*, 1941, 17: 469-482

ROGERS MEMORIAL SANITARIUM

OCONOMOWOC, WISCONSIN

A PRIVATE Sanitarium (incorporated not-for-profit) *for the treatment of*

Diseases of the Nervous System

All approved methods of therapy are used

MEDICAL STAFF

OWEN C. CLARK, M.D.
Medical Director

C. H. FEASLER, M.D.

JAMES C. HASSALL, M.D.
Consultant

TUCKER HOSPITAL, Inc.

212 West Franklin Street, Richmond, Virginia

A private hospital accepting for diagnosis and treatment organic neurological conditions, selected psychiatric and alcoholic cases, metabolic disturbances of an endocrine nature, individuals who are having difficulty with their personality adjustments, and children with behavior problems. Patients with general medical disorders admitted for treatment under our staff of visiting physicians.

Under the Professional Charge of

Dr. Howard R. Masters, Dr. James Asa Shield and Associates

North Shore Health Resort

on the shores of Lake Michigan

WINNETKA, ILLINOIS

NERVOUS and MENTAL DISORDERS
ALCOHOLISM and DRUG ADDICTION

Modern Methods of Treatment

MODERATE RATES

Established 1901
Licensed by State of Illinois

Fully Approved by the
American College of Surgeons

SAMUEL LIEBMAN, M.S., M.D.
Director

225 Sheridan Road

Winnetka 6-0211